INFLUENCE OF PERIPHERAL AND CENTRAL INSULIN ON SYMPATHETIC NERVOUS SYSTEM ACTIVITY. Martin Muntzel and Alan Kim Johnson. Depts. of Psych. and Pharmacol. and the Cardiovasc. Cent., Univ. of Iowa, Iowa City, IA, 52242.

While the role of insulin in the regulation of adiposity and energy balance is well established, recent studies examining physiological mechanisms of satiety indicate that insulin may also communicate changes in energy status to the central nervous system (CNS). These studies support the hypothesis that insulin operates in a negative feedback loop, whereby high post-prandial insulin levels activate specific binding sites in the hypothalamus to trigger satiety signals and to decrease food intake. An additional role for central insulin as a regulator of sympathetic function has emerged from experiments examining mechanisms of hypertension. Correlational studies have demonstrated strong positive relationships between obesity, insulin resistance, hyperinsulinemia, and hypertension. Insulin may elevate blood pressure through an activation of the sympathetic nervous system (SNS). Although most authors agree that insulin acts within the CNS to increase SNS activity, the mechanism and anatomical sites remain to be elucidated. To determine whether intraventricular insulin elicits excitation of the peripheral SNS, we infused insulin (10 μU/min) or vehicle into the third ventricle of chloralose-anesthetized rats while recording multifiber activity from the lumbar sympathetic nerves (LSN). Both LSN activity and blood pressure were increased by insulin whereas heart rate and blood glucose concentrations were not altered by the procedure. Our present studies are using brain lesion techniques to identify specific brain sites responsible for sympathoexcititory effects of central insulin.

Source: https://www.industrydocuments.ucsf.edu/docs/lnfb0000